count rising parallel with the temperature curve, the myelocytes decreasing proportionately. The chart on preceding page elucidates this more graphically.

This rapid increase in the number of polymorphonuclears suggests an intercurrent leucocytosis. Between the first and last observation made in our case the proportion of white to red corpuscles oscillated from 1:18 to 1:27. Taking normal variations and similar factors into consideration, this difference in the ratio can be discarded. An actual decrease in the myelocytes, however, can be noted, the afebrile count showing a reduction of from 53.2 per cent. to 44.7 per cent., while the febrile count also shows a drop from 31.2 per cent. to 30.5 per cent.

I desire here to express my thanks to Drs. Louis Heitzmann and M. S. Kakels for control counts.

A CASE OF CERVICAL AND BULBAR TABES, WITH NECROPSY.[1]

By Solomon Solis Cohen, M.D.,
of Philadelphia,

and

William G. Spiller, M.D.,
of Philadelphia.

Clinical Report by Dr. Cohen.

The case that forms the subject of the present paper was reported to the Philadelphia Neurological Society in 1889 as a case of "Bulbar Paralysis, with Marked Disturbances of Pain and Temperature Senses and Other Phenomena Pointing to Syringomyelia," and the report was published, together with remarks by Dr. Charles K. Mills, who had seen the patient in consultation, in the *Medical and Surgical Reporter* for July 13, 1889, p. 34 *et seq.* Despite the caution observed by Dr. Mills and myself in the title of our report as just quoted, abstractors and authors have referred to the case as one of undoubted syringomyelia with bulbar symptoms, and as such it has entered into, and to that extent vitiated, the statistics of the affection in question. The results of the necropsy and of Dr. Spiller's careful study of the brain and cord show that the disease causing the somewhat unusual phenomena reported was a rare form of tabes, and not syringomyelia. It is to be hoped that this supplementary record will at least obtain sufficient reading to prevent future incorrect references.

Following Dr. Spiller's suggestion I shall repeat almost in full the previous report, as the symptomatology does not readily lend itself to further condensation and is of great importance. In 1889 the following description was given:

---

[1] Presented at the meeting of the American Neurological Association, June, 1901.

The patient is a man, aged fifty-five years, tall, large framed, and of powerful build. He has weighed 230 pounds, and has been capable of feats of great muscular strength. Thirty years ago he contracted the initial lesion of syphilis. He has had secondary but not tertiary symptoms. He cannot give the exact date of the onset of his disease, which probably developed insidiously. In 1872, however, he had attacks of nocturnal incontinence of urine, following relief of a stricture by dilatation and internal urethrotomy, and this symptom has persisted more or less ever since. In 1878 or 1879 his right eye gradually became affected with a peculiar form of ptosis. The lid is closed, but it is possible for him to open it and to keep it open. He finds it more comfortable closed, and hence ordinarily keeps it so. The pupil, he states, was contracted at the time referred to, that of the left eye being dilated. Some years ago, suddenly and without apparent cause, the left pupil contracted and the right pupil dilated. In 1886, being annoyed by a constant biting of his cheeks, he had several teeth extracted. He felt no pain upon their removal. Since that time the present condition has gradually developed. The symptoms now presented may best be described in groups, as they affect motion, sensation, special senses, and organic functions, including nutrition.

Motion. The right side of the face is partially paralyzed. The eye is closed—that is to say, there is apparent ptosis. It is only apparent, however, for the patient can raise the lid at will and does so to do any work. He keeps the eye closed, however, when reading or writing. He can open and close each eye independently of the other. He has very nearly perfect motion of the eyeball, but there is some slight impairment in the internal rectus and in the superior oblique—not noticeable, however, except upon careful testing. The pupil is slightly dilated, but responds at times both to light and accommodation. When I first saw him, a year ago, it was almost completely dilated; a week or two afterward it became contracted; a little later it again dilated to its present condition, its diameter being larger than that of its fellow. The forehead, in elevation of the brows or in frowning, wrinkles nearly as much upon one side as the other, there being an equal number of lines, but somewhat less distinct in the nasal half of the right side. The fan-shaped lines around the eye are nearly as distinct upon the right as upon the opposite side. Below the eye there is a more marked paralysis. The labio-nasal fold is not so prominent as on the other side, but there is no drawing of the mouth when at rest. The lips apparently move equally on both sides in smiling and talking, but the patient cannot whistle, and in the attempt to pucker the lips a somewhat triangular aperture with the apex pointing downward and outward appears at the right side. Prehension and suction are difficult, so that he has gradually been compelled to reduce, and finally almost to abandon smoking. Mastication is quite difficult, and very imperfectly performed. There is considerable difficulty in deglutition, so that solid foods must be washed down with water. Saliva accumulates to a great extent, on account of the difficulty in swallowing it; it can be ejected, but there is some impairment in this action also. There is a sucking-in or drawing-in of the cheeks, probably due to a tendency in the lower jaw to fall slightly, and of the cheeks to fall into the space between the upper and lower teeth. The tongue exhibits fibrillary tremors. It is protruded to the left, though it can be moved in all directions. There is hemiatrophy, the left side being the smaller. The difference between the two sides is not so marked at present as when the case first came under observation—that is to say, the right side has undergone partial atrophy as well. The various motions of the tongue can be performed, though with some difficulty. There is no paralysis of motion of the palate or of the larynx. Speech is somewhat thick, the greatest difficulty in articulation affecting the labial and sibilant sounds. It has improved slightly under treatment. The patient's grip is good, his power of locomotion unimpaired, and he is not conscious of any paralysis of the limbs. However, the right leg at times feels somewhat heavy; and he admits he is not able to lift as heavy weights or to walk as great distances without fatigue as formerly.

Sensation. This varies from day to day, but the following are the principal constant conditions: Tactile sense in general is almost perfectly preserved; at time, there are places upon the face, lips, and within the throat where it seems blunted. The temperature sense is both blunted and perverted upon the right side of the face, more markedly below the eye, where cold is at times called warm. On the trunk and limbs the temperature sense is preserved, but the patient experiences a peculiar feeling of faintness upon the application of cold water, so that he cannot use it for bathing or washing. He has had to abandon sea-bathing for this reason. He could wade into the sea until the water reached the upper third of the thighs, but beyond that it would cause this peculiar feeling of shock with sense of impending dissolution. The pain sense is peculiarly blunted; it is absolutely gone so far as sharp points are concerned in the right side of the face, on the neck, and in irregular areas over the trunk and thighs. These, so far as observed, do not appear to be in relation to particular nerve distribution, but the patient could not submit to the very prolonged and tedious examinations necessary to accurately define the limits of the analgesic areas, which affect both sides of the trunk, more especially above the line of the nipples, both arms and both thighs. To the continued electric current, sensation is perverted. Upon the left side of the face galvano-cutaneous sensibility is almost completely absent; but there are a few points, especially in the canine fossa, where it is so acute as to be absolutely painful. Sudden interruptions of the current are felt as blows referred to the cathode, even upon places where the tingling of the uninterrupted current is not felt. Taste and light sensations can be awakened by interruption of strong currents. Sensation to the faradic current seems preserved at all points, though perhaps quantitatively diminished. Itching of the eyelids, especially of the right side, is frequent. Occasionally neuralgic pains affect the legs, especially the right, and sometimes there are griping pains in the abdomen, not due to indiscretion in food or ordinary causes. There has never been headache. At times there is overpowering drowsiness; the head drops forward, and there is a sensation as of cold water being poured over the neck, followed by intense heat.

Special Senses. Hearing is unimpaired. Smell is somewhat impaired. Taste is absent from the anterior portion of the tongue, but upon pressing the sapid substance—salt or sugar—against the hard palate with the tongue, taste is recognized. The patient says that he detects flavors of food swallowed. Sight is much impaired. No ophthalmoscopic lesion was discovered by Dr. Jackson, who, indeed, found vision exceedingly good. Dr. Jackson gives the following notes of the condition of the eyes:

"Mr. X. was first seen with Dr. Cohen, February 14, 1888, on account of an irritable ulcer of the cornea, which, under the use of eserine and applications of hot water, had quite healed ten days later. At this time it was noted that the pupils were small and reacted very slowly and imperfectly. The patient again came under observation, August 31, 1888, and was seen several times in the succeeding three months. During this period there was no material change in the condition of his eyes, which is as follows: He complained of inability to read or use his eyes in business, and the right eye was constantly kept closed by complete relaxation of the elevator of the upper lid, without any contraction of the orbicularis. Nevertheless, when requested to do so, he opened his eye fully without evidence or sense of excessive effort, and kept it open as long as desired for purposes of examination.

"Both eyes presented compound hyperopic astigmatism, requiring for its correction:

R. + 1.50 sph. + 1.25 cy. axis 120, giving V. = 5/v partly.
L. + 2 sph. + 0.62 cy. axis 90, giving V. = 5/iv most letters.

"His color perception was normal.[1] Fields of vision for form and colors were normal or very slightly contracted concentrically; that is, they were a little smaller than the average normal field, but of normal shape. The ophthalmoscope showed that the optic disks were rather anæmic and 'suspiciously gray,' but they could not be pronounced positively abnormal. Fundus otherwise normal. Media clear.

"The pupils were unequal. Right circular, 6.5 millimetres in diameter; left slightly ovoid with long axis vertical, measuring 4.5 mm. These were the dimensions of the pupils with the visual axes about parallel. There was no power of accommodation, but with strong convergence the pupils very slowly contracted to right 5 mm., left 3.5 mm., and upon relaxing the convergence they very slowly dilated to their original size. They gave no reaction whatever to light.

"The extra-ocular muscles showed power of abduction 10°, adduction 30°, sursumduction sometimes as high as 6°, sometimes as low as 1°. Vertical diplopia produced by a prism with its base up or down showed exophoria (tendency for the visual axes to diverge), varying from nothing to 5° when looking at an object directly in front. A red glass placed before one eye caused crossed diplopia, which increased as the eyes were turned toward the right, and disappeared on looking to the left (special weakness of the right external rectus). With horizontal diplopia, produced by a prism so placed as to cause a horizontal displacement of the image on one retina, the visual axes sometimes remained on the same level; sometimes the right was directed higher than the left, sometimes the left higher than the right (right or left hyperophoria). Without the use of the red glass, or some such intervention, there is no squint and no diplopia, but he gives a history of diplopia in other years."

The patient states that he cannot read or write for long periods, on account of failure of vision, and on account of dimness he has recently been compelled to intermit his occupation in the machine and carpenter shop he had fitted up in his home to employ his time. Within a few weeks he has occasionally experienced sudden attacks of transient blindness, accompanied with vertigo. He has had subjective vertigo, without visual disturbance occasionally, for many years. He has never fallen or lost consciousness in any of these attacks. There is no other visual disturbance.

---

[1] But the tones perceived by the right eye were always somewhat different in intensity from those perceived by the left. White at times appeared slightly "yellowish-red" to the right eye; but it could, nevertheless, detect a very faint blue.—S. S. C.

Organic Functions. The heart's action is feeble and irregular. The heart is slightly dilated, but not sufficiently so to account for the disturbance of its rhythm or the enfeeblement of its contractions. At times there are attacks of palpitation. The pulse is soft and will average some eighty odd beats in the minute. There is rarely intermittence. Respiration is free and regular, and normal in its acoustic phenomena. Dyspnœa upon exertion is recent and inconstant. Digestion is not perfect, but offers no special neurotic phenomena. The appetite is failing, and assimilation does not seem to be good. Urination is at times difficult, at times involuntary. Incontinence occurs only at night, at irregular intervals, varying in duration from one to three or four nights. It is a frequent symptom. When there is no incontinence the urine is, as a rule, scanty, varying in quantity from sixteen to forty fluidounces per diem. It is of high color, odor, and specific gravity, containing an excess of urates and phosphates, some calcium oxalate, abundant mucus and epithelium, but no casts, albumin, or sugar. Defecation is irregular, constipation alternating with diarrhœa, the latter often being difficult to control. Recently there have been involuntary, but never unconscious, movements of the bowels at night. Sexual power has gradually become impaired, and has for some months been lost. Nutrition is evidently much impaired. There has been a loss of sixty pounds in weight in six months. The loss has been oscillatory, but, on the whole, progressive. It is more than can be accounted for by difficulty of deglutition, and has occurred despite special attention to the digestion, and the continuous use of highly concentrated and predigested aliment. The muscles are flabby, and, while of good bulk for a less massively designed person, are evidently wasted from former conditions. There are no fibrillary tremors.

Reflexes. The knee-jerk and muscle-jerk are well preserved. The palate reflex is good. The presence of an electrode on the base of the tongue, or in the œsophagus or larynx, induces excessive gagging, even when used merely as a sound; and this becomes intense upon passage of the weakest current, the latter being extremely painful. Tactile sensation in these regions varies, but is apparently somewhat perverted, and the pain and temperature senses are blunted but not lost.

Mentality. The intellectual function is good. There is some disposition to repetition of old stories, and a forgetfulness of details, but the patient being called upon to bring order out of the complicated accounts of a relative's insolvent estate, was able to do so with great ease and clearness of judgment. There are occasional alarming dreams, but probably no more than anyone might have.

Co-ordination is apparently perfect. The patient can execute complicated movements with precision, can stand with his eyes closed, and can balance himself on one foot.

Electrical Reactions. All the muscles respond to a faradic current, though with greatly diminished activity, especially on the right side of the face. Sensation to the faradic current is preserved, but blunted. To the galvanic current response is good in the limbs, but much diminished in the face on both sides, the right more markedly. There are no degenerative reactions. The current produces marked hyperæmia of the part to which the cathode is applied. The patient, during the winter, would frequently complain, on entering the office, that his nose was icy cold, and it would appear blanched, and have a sensation of coldness to the touch. The constant current of a strength of from 2 to 4 milliampères would, in a few minutes, restore the color and warmth. This (cathodal) application was excessively painful.

While the question of diagnosis, and not that of treatment, was the one to be discussed at the meeting to which the foregoing report was made, it was thought well to say that the electric applications appeared to have kept up the nutrition of the facial muscles to a degree greater than could have been maintained otherwise. There had been considerable improvement in the motion of the brow, and in articulation, and the face had not wasted to the same degree as the body generally. General galvanism had also been employed, but not so thoroughly as the facial applications. Digitalis and strophanthin for the heart, in alternation; strychnine constantly for general nutrition; and at different times arsenic, mercuric chloride, auro-sodium chloride, or silver nitrate in the attempt to hold in check the neural degeneration; with temporary expedients to assist digestion, facilitate urination, and repress unpleasant symptoms of various kinds, had been employed after it had been demonstrated that mercury specifically and potassium iodide were alike useless. Malt, alcohol, coca, and the most easily assimilable and concentrated articles of food, including beef peptonoids, peptonized milk, etc., constituted the diet.

The patient lived for nearly twelve years after this report, dying in 1900 from pulmonary œdema consequent upon innutrition, following complete paralysis of deglutition. The history during this period was one of progressive but slow emaciation and loss of strength generally and in both extremities, with decided atrophy of the hands, especially of the left hand. The complaints of "sucking-in of the cheeks" increased, but prehension and suction still remained sufficiently good to permit both pipe and cigar to be enjoyed at various times. There was much irregularity as to this, as also in regard to distinctness or thickness of articulation. Taste was largely lost, and common sensation in the mouth and tongue became so poor that the patient would often lose his food in his mouth and have to find it and push it into the grasp of the pharyngeal muscles with his fingers. For this reason he took only such food as could be swallowed in large morsels, with little chewing or buccal manipulation. The tongue could be moved readily, but was atrophied apparently equally on both sides. There was considerable pain along the ulnar surface of the right hand, and the patient always bent the little and ring fingers out of the way in shaking hands on account of the pain caused by pressure upon them. He also began to complain greatly of pain in the abdomen, and any accidental touch upon it caused great distress. The darting pains down the right leg occurred less frequently, but with greater severity, and morphine or one of its succedanea was necessary at times to give relief. The abdominal pain could not be relieved by internal medication, but the patient was able to manipulate his own abdomen with various analgesic oils, or to place upon it warm cloths and the like. These applications were frequently followed by relief for prolonged periods. It was evident, and it was stated to the patient, who had himself studied medicine although he had never practised, that the pain was not local but due to involvement of the nerve roots. Notwithstanding this he found relief in the local application, whether by physical action, reflex or local, or by psychical action. Mentality continued excellent, however, to the last.

There were during the last years a few transient attacks of vertigo; never a gastric crisis; never ataxia or abasia. He had two attacks of uræmia—one about two years before death, and one about ten days preceding the fatal illness. The former seemed to have been superinduced by relatively excessive doses of heroin, self-administered, to relieve unusually severe attacks of pain. The quantity taken was one eighth of a grain every hour for some four or six hours. Unconsciousness, however, was not caused by cessation of renal secretion, but apparently by the drug, as it occurred after the morning evacuation of the bladder, which, when I saw the patient, was nevertheless over-distended, and from which nearly twenty ounces were removed by catheter. Secretion, however, after this became deficient, almost absent, and death was apparently imminent. Under treatment by persistent hypodermoclysis with physiological saline solution, the hot air bath, and the free use, hypodermically, of nitroglycerin, spartein, strychnine, musk, and camphor, consciousness was regained in about seventy-two hours and was followed by recovery. Convalescence was slow. A catheter urethritis developed, which, however, yielded to appropriate local measures and internal medication.

After this some enfeeblement of memory was noticed, though perhaps not more than the age of the patient might account for. He had to make memoranda of matters to be attended to or inquired about, but that gave him sufficient clue to start the train of thought going. His reasoning powers were still clear; he preserved his interest in books and affairs, and continued to potter around his workshop. He complained, however, that he could not continue at work very long without fatigue, and especially that the pressure of the handle of instruments upon the palm was painful. His writing was good. His chief anxiety arose from progressive failure of vision. Dr. Schneidemann from time to time examined the eyes, and favors me with the following abstract from his books of the more important data:

"Mr. X. was first seen by me January 14, 1896, in Dr. Jackson's absence, he being a patient of Dr. Jackson. From May, 1896, to June, 1898, Dr. Jackson was in the city, and I did not see the patient. On October 17, 1898, he consulted me and once again early in 1899. May, 1899, he was seen by Dr. Jackson. He again consulted me for the last time, October 31, 1900. His chief complaint in all of my interviews with him was irritation of the conjunctiva, such as smarting, scratching, burning, etc. In fact, his principal object in coming was to endeavor to obtain relief from these symptoms. Certain abnormal ocular conditions were present when I first saw him, which persisted with slight changes.

"The right eye presented the appearance of pretty complete ptosis. He was, however, able to raise the lid by voluntary effort, but only did so for some special purpose when asked to, but not ordinarily, as for the purpose of using the eye in conjunction with its fellow for vision. The left eye presented no anomaly of the muscles of the lid.

"The refraction was hypermetropia with astigmatism in both eyes, which varied but slightly at different examinations. October 31, 1900, I prescribed –

O.D. + 3.50 ⊂ + 1. cy. ax. 125
O.S. + 4. ⊂ + 0.62 cy. ax. 90

for near use, the distance correction being:

O.D. + 1.50 ⊂ + 1. cy. ax. 125 = 4/8.
O.S. + 2. ⊂ + 0.62 cy. ax. 90 = 4/5 +.

"The pupils were irregular, their size variable, the right constantly larger than the left. Light reaction was absent at each examination, but contraction was present upon convergence of the visual axes (Argyll-Robertson pupil). At the examination first made by me (1896, etc.) the posterior media were found to be clear. In January, 1899, there was some beginning cortical opacity of the lens of the right eye. This continued to progress so that when I saw him again, October, 1900, the patient himself recognized that the vision in that eye was failing. It did not, however, fall below 4/8, as above mentioned. At this time the lens of the left eye began to show some disturbance.

"The fundi never exhibited any positively significant lesions. The fields of vision for form and color were normal. The nerves did not present any marked departure from the normal, although a suspicion was entertained at times that the color was rather pale; crescents of partial choroidal atrophy were present, but not more marked than is often the case.

"A marked degree of exophoria could be brought out, increasing as the patient looked to the right, and almost diminishing to 0 as he looked to the left; also, very slight right hyperphoria on looking to the right, none on looking to the left. Abduction was 10, and adduction 30 centrads."

The attack of uræmia about ten days before death followed a chill consequent upon exposure in an attempt to assist a carpenter who was repairing the windows in the machine shop. Recovery from the suppression of urine, and the unconsciousness attendant upon it, was more prompt than upon the previous occasion, though the attack was followed by considerable weakness and a greater loss of memory was apparent. Indeed, there was a partial aphasia—that is to say, the patient utterly failed to recall the particular word desired, though perhaps that same word would come readily at another time. He was, however, convalescent and moving around the room, when suddenly one night his utterances became so indistinct that he could not be understood by those about him, and when seen the next morning there was considerable paralysis of the left side of the face and throat. Swallowing was impossible. Articulation was impossible unless the jaw was held shut, when some motion of the lips could be made, and a few words made out. Once or twice during the attack this condition passed off temporarily, so that he could speak one or two words in answer to questions—a fact by which we proved that he had preserved consciousness and was not in coma. He could move his hands and feet, and the paralysis seemed to be chiefly in the muscles closing the jaw and the palate and pharynx. He could even protrude the tongue with some difficulty. As from thirty to sixty ounces of urine were passed daily, and as the heart's action and respiration kept up very well, this last phase of the illness could not be attributed to uræmia or to cardiac failure. This condition lasted nearly four days. The breathing became labored, and the temperature elevated, about twelve hours before death, during which period there was unconsciousness. Death took place apparently from œdema of the lungs. As neither food, water, nor medicine was given by the mouth, there was no possibility of "deglutition pneumonia." The saliva ran out of the mouth at first, but toward the last secretion ceased.

At the autopsy made by Dr. David Riesman, in the presence of Dr. Charles K. Mills, Dr. A. A. Eshner and myself, examination of the thorax and abdomen was not permitted, but we were allowed to remove the brain and spinal cord. The description of the external appearance of the cadaver and the macroscopic aspects of the brain and cord, as dictated by Dr. Mills, follows:

"October 21, 1900. Body of a normal white man, about middle height. Rigor mortis intense. Superficial abrasion on the inner side of the left thigh. Some red spots on the right leg below and over the knee. Face has a peculiar, bluish-gray color (argyria). Nipples also present a bluish-gray color, added to the natural brown. Marked atrophy of the interosseous muscles, and also of the extensor minimi digiti, especially on the left. Hands seemed flexed, thumbs abducted (rigor mortis). Nail-beds bluish-gray in color, especially at base. Body has been injected. Cup-marks from the first to the fourth interspace on both sides. Marked atrophy of the left arm and forearm, particularly the forearm. Left thigh also atrophic, but not markedly so. Marked atrophy of the left calf-muscles. No atrophy of the muscles of the foot.

"Scalp thin. Skull thinner than normal generally. Dura not adherent. No excess of fluid. Fissures deep. Pia arachnoid œdematous and somewhat opaque. Basilar artery exceedingly long and greatly dilated throughout its course. In whole motor region (fronto-Rolandic) from posterior central convolution forward to precentral sulcus, the convolutions are atrophied and the general area presents a depression. The sulci are usually deep and wide, particularly the two limbs of the fissure of Sylvius. The posterior portion of the third frontal is also involved (the whole of operculum and some surrounding tissue), and the foot of the third frontal. On the right side is a recent meningeal hemorrhage at the upper portion of the ascending frontal convolution. There is a marked depression in the situation of the first frontal convolution on the right, forming a shallow trench, oval in shape, the size of a thumb, near the median edge of the hemisphere. The anterior end of the depression is about one inch from the tip of the hemisphere, and the posterior end is near the anterior extremity of the calloso-marginal fissure. There is a depression also on the median aspect, of the same size, immediately posterior to the one on the lateral aspect, extending from the gyrus fornicatus to the edge of the hemisphere. On cutting off the occipital lobes by Dejerine's method, a marked difference appears in the transverse section of the exposed lateral ventricles, the left being nearly twice as large as the right, and oval rather than circular. The choroid plexuses are dark. The left lateral ventricle (on making sections at the level of the callosum) is apparently somewhat larger than the right. The difference in the previous section is due probably rather to dilatation of the posterior horn than to that of the ventricle itself."

After the report of this case to the Neurological Society the diagnostic interest to the attendant gradually faded, becoming replaced by the human interest. Scientific observations and records were not systematically made, attention being given rather to the conservation of strength and the palliation of symptoms. Whether or not therapeutic measures which were continued practically as reported in 1889 are to be given credit for the slow progress of the case it is impossible to say, because the long duration of symptoms before the patient came under observation showed that, for some reason, the tendency to extension was not active. It is possible that closer study during the terminal years might have revealed during life the exact nature of the case, but the diagnosis, ante-mortem, proceeded no further than to postulate a slowly progressive degenerative lesion of irregular distribution, principally affecting the bulbar and upper cervical region, bilateral, though chiefly affecting the right side, and involving the posterior nerve roots at various levels of the cord. Dr. Spiller's careful and able study and analysis will show in how far this was correct, and wherein it failed.

MICROSCOPICAL STUDY AND COMMENTS BY DR. SPILLER.

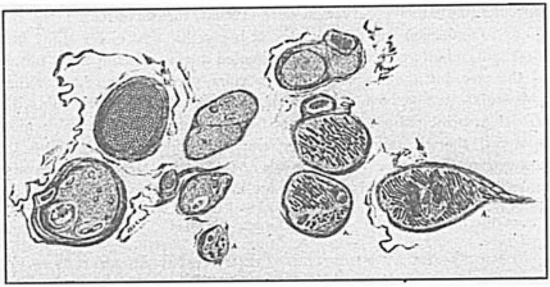
(From the William Pepper Clinical Laboratory, Phebe A. Hearst Foundation.)

No distinct degeneration is found in the posterior columns in the mid-lumbar region. The nerve fibres along the posterior septum are not quite so numerous as elsewhere in the posterior columns, but no positive degeneration is found even here. Some of the bundles in the extramedullary portion of the posterior roots show a slight degeneration, but the medullated nerve fibres passing into the posterior horns are numerous. It is doubtful whether the anterior roots could be regarded as at all degenerated. When the lumbar posterior roots are collected and cut separately from the cord so that a large number may be obtained in transverse sections, very little degeneration of these roots is seen. The nerve cell bodies in the anterior horns are numerous and are apparently normal.

In the mid-thoracic region there is no pronounced degeneration of the postero-median columns. The nerve fibres possibly are not quite so numerous along the posterior septum as in a normal cord, but the diminution in the number of the fibres, if it exists, is very slight. When a large number of posterior roots are cut separately from the cord they are found to be much degenerated, and some of the bundles contain no medullary nerve fibres at all, and others very few such fibres. (See Fig. 1.) Even macroscopically the atrophy of the posterior roots of this region was very perceptible when the spinal cord was examined. The bloodvessels within the degenerated posterior roots have much thickened walls and are of a glassy, homogeneous appearance when stained with ammonium carmine. A slight area of sclerosis is seen on the median side of each posterior horn in the area of the root entrance zone.

In the lowest cervical region, at what is doubtless the eighth segment, the posterior columns appear to be normal except in a narrow band within the column of Burdach along each postero-lateral septum and in the right root entrance zone. The left root entrance zone appears to be almost normal. This narrow band of slight degeneration along each postero-lateral septum is the result of partial degeneration of posterior roots in the upper thoracic region. The root entrance zone on the right side in the eighth cervical segment is much degenerated, as is also a portion of the extramedullary posterior root of this segment.

FIG. 1.



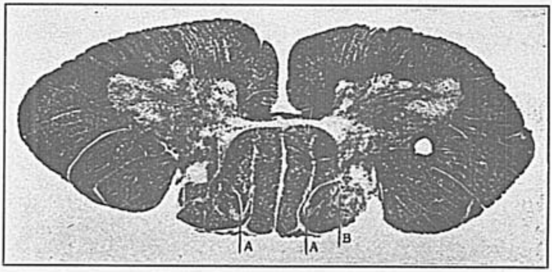
Posterior roots of the mid-thoracic region, cut transversely. The bundles A, A, A, A are much degenerated. The nerve fibres have entirely disappeared in the other bundles.

At the cervico-thoracic junction—*i. e.*, a little lower—the posterior roots in their extramedullary portions are intensely degenerated. At the eighth cervical segment the posterior root on the left side is almost normal. On the right side the posterior roots in the lower cervical segments (see Fig. 2) are partially degenerated, and fewer medullary fibres are found entering the right posterior horn than the left ; at the mid-cervical region no degeneration even of the right posterior roots is seen. A small but distinct area of degeneration in the lower cervical region above the eighth segment is found in each column of Burdach near the periphery of the cord and adjoining the postero-lateral septum, and is in the same position as in the eighth cervical segment. It is the result of degeneration of upper thoracic posterior roots. The degeneration of the posterior roots in the cervical region is almost confined to the right side and the lower cervical levels. No recent degeneration is detected by the Marchi method in sections from the lower cervical region, and the process was evidently not acute. The anterior roots are normal.

No distinct round-cell infiltration of the pia of the cord is present. The nerve cell bodies in the anterior horns are numerous and highly pigmented, which is not extraordinary in consideration of the age of the patient.

Sections from the lower part of the medulla oblongata show no degeneration with Marchi's stain. The nucleus of the left twelfth nerve in its inferior portion is much smaller than the corresponding portion of the right twelfth nucleus (see Fig. 3), and contains fewer nerve cell bodies and fewer medullated nerve fibres. The intramedullary nerve fibres of the left twelfth nerve are not so numerous as those of the right in the inferior portion of the medulla oblongata, but at higher levels a distinct difference between the two nuclei and the intramedullary fibres of the twelfth nerves cannot be observed, and there is no distinct degeneration of these parts. The extramedullary portion of each twelfth nerve is normal.

FIG. 2.



Photograph of a transverse section of the spinal cord from the lower cervical region. A, A. Narrow band of degeneration along each postero-lateral septum, the result of partial degeneration of posterior roots in the upper thoracic region. B. Degenerated root entrance zone on the right side. The corresponding zone on the left side is not degenerated.

The posterior nucleus of the tenth nerve is normal on each side. It is difficult to determine a moderate degeneration of the nucleus ambiguus, but on neither side is this nucleus distinctly degenerated. Some of the nerve cell bodies in this nucleus on each side are much pigmented. The fasciculus solitarius on the left side is greatly degenerated, but on the right side this fasciculus is normal. (See Fig. 3.)

Along the floor of the fourth ventricle are proliferations of neuroglia forming irregular projections into the ventricle.

The lemniscus is normal.

There is some slight perivascular round-cell infiltration in the pia and within the medulla oblongata, but this is not important.

The seventh nucleus and intramedullary fibres of the seventh nerve on each side are normal. The extramedullary portion of the left seventh nerve is probably slightly degenerated, but sections from a corresponding portion of the right seventh nerve appear to be normal.

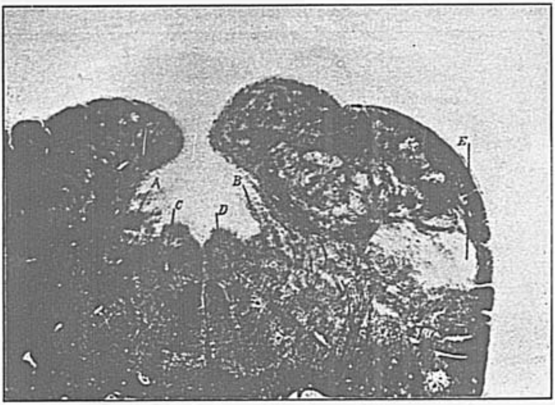
The nucleus and intramedullary fibres of each sixth nerve appear to be normal. The right sixth nerve in its extramedullary portion is partially degenerated, while the left sixth nerve in the corresponding portion is apparently normal.

In the sections examined the fifth motor and sensory nuclei are present only on one side, and these nuclei are normal. A transverse section through the extramedullary portion of the left fifth nerve near the pons shows considerable degeneration of the nerve roots. The spinal root of the fifth nerve on each side is much degenerated, and possibly the ventral portion is a little more degenerated than the dorsal in the inferior part of the medulla oblongata.

The right cerebral peduncle is considerably smaller than the left.

Sections through the oculomotor nuclei contain many nerve cell bodies in each lateral nucleus and in the median nucleus, but the nerve cell bodies in the left lateral nucleus are distinctly fewer than in the right, and cell bodies in all parts of the oculomotor nuclei are atrophied, deeply pigmented, and have peripherally situated nuclei. The contrast is striking in comparison with nerve cell bodies of an oculomotor nucleus believed to be normal.

FIG. 3.



Photograph of a section from the lower part of the medulla oblongata. The nucleus of the left twelfth nerve (D) is much smaller than that of the right twelfth nerve (C) at this level. The descending root of the ninth and tenth nerves on the left side (B) is degenerated, while that on the right side (A) is normal. The spinal root of the fifth nerve (E) is degenerated on each side, but not so completely as the photograph represents, as under the microscope it contains a few nerve fibres.

The giant-cells of the left paracentral lobule are normal, but in some portions of the cortex from this part the capillaries are very numerous.

The right optic nerve appears to be normal, but the left is partly degenerated.

The important findings in this case are: Degeneration of the posterior roots of the spinal cord, hardly perceptible in the lumbar and lower thoracic regions, very distinct in the middle and upper thoracic and on the right side in the lower cervical region but absent in the middle and upper cervical regions. No distinct degeneration of the columns of Goll, and very slight and limited degeneration of the columns of Burdach in the upper thoracic and cervical region; atrophy of the inferior part of the left twelfth nucleus; integrity of the extramedullary roots of the twelfth nerves; intense degeneration of the left descending root of the ninth and tenth nerves, possibly a slight degeneration of the extramedullary portion of the left seventh nerve near its exit from the medulla oblongata; partial degeneration of the extramedullary portion of the right sixth nerve; intense degeneration of the spinal root of each fifth nerve, with partial degeneration of the extramedullary portion of at least one fifth nerve; atrophy and changes in the nuclei of the oculomotor nerves, resembling those occurring when the axones are diseased.

These lesions justify the diagnosis of an atypical form of tabes, and the data of importance in the clinical history are: The acquisition of syphilis some years before the appearance of symptoms of nervous disease; attacks of nocturnal incontinence of urine beginning in 1872; a peculiar form of ptosis of the right eye beginning in 1878 or 1879—*i. e.*, a drooping of the right upper lid, which might have been the result of partial paralysis of the levator palpebræ superioris or of loss of the muscular sense in the lid. The lid could be raised voluntarily, but was more comfortable closed—"springing pupils" dating from 1878 or 1879; it is stated that the contraction and dilatation of the pupil in each eye varied from time to time; some weakness of the facial muscles which may have been the result of loss of muscular sense in the face, as it seems that co-ordinated movements, such as whistling, were most affected; difficulty of mastication and of deglutition; atrophy of the tongue, noticed at first on the left side; disturbance of sensation, especially for temperature and pain, and especially on the right side of the face, which seems to suggest that the right-sided ptosis may have been of centripetal rather than centrifugal origin—*i. e.*, from implication of the muscular sense; occasionally griping pains in the abdomen, not due to indiscretion in food or ordinary causes; and neuralgic pains in the lower limbs, these seeming to be the lancinating pains of tabes; a "suspicious grayness" of the optic disk, inequality of the pupils, loss of reaction of the irides to light, loss of power of accommodation, weakness of the right external rectus muscle, failure of vision—all observed by Dr. Jackson in 1888—loss of sexual power.

In addition to these symptoms atrophy of the upper limbs and pain in the ulnar distribution of the right hand developed, the latter explicable by the degeneration of the posterior roots in the lower cervical and upper thoracic regions on the right side.

It is remarkable that this man had no inco-ordination in the movements of the upper limbs, and that he had not is very evident from a specimen of his handwriting. Dr. Cohen says he had no distinct inco-ordination, and was able to write well and clearly and to do work in his carpenter shop up to within a few weeks of his death. The absence of ataxia is explained by the fact that in the cervical region only the lowest posterior roots were degenerated, more especially those on the right side, and even here these roots contained many normal fibres. The degeneration of the posterior roots in the upper thoracic region explains the severe pains extending into the abdomen.

Dr. Cohen, in his paper published eleven years before the death of the patient in 1900, remarked that he believed an irregularly diffuse and slowly progressive degenerative lesion existed, affecting principally the medulla oblongata. The microscopical examination of the tissues shows that this opinion was correct. The slowness in the development of this atypical form of tabes was most extraordinary, for even after the disease had existed many years, presumably at least since 1878 or 1879, the degeneration of the posterior roots was almost confined to the upper thoracic and right lower cervical regions, and was not complete in these portions. The degeneration of the posterior columns was slight, ~~and was~~ partially masked by the presence of normal fibres from lower levels of the spinal cord. The sections are totally unlike those of the ordinary form of tabes, as the columns of Goll are nearly or fully intact.

It is exceedingly important to note that when this case was reported by Dr. Cohen, in 1889, and at a time when the symptoms of the disease were very pronounced, the knee-jerks were well preserved. The explanation is found in the almost complete integrity of the posterior roots of the lumbar region. The knee-jerks in cervical tabes have usually been found absent.

The mentality in Dr. Cohen's patient does not seem to have been fully normal, because it is stated that there was some disposition to repetition of old stories and a forgetfulness of details. Marie[1] has said that in bulbar tabes mental disturbance is not uncommon, and, indeed, it is not very uncommon in the ordinary lumbar form of tabes.

In 1889 co-ordination was said to be perfect, the patient could execute complicated movements with precision, could stand with the eyes closed, and could balance himself on one foot. As Dr. C. K. Mills pointed out at that time, the symptoms were those of bulbar paralysis, but yet the case was unlike one of bulbar paralysis on account of the marked changes in the pain and temperature senses below the neck as well as above it; the involvement of the bladder, bowels, and sexual organs; the general wasting, and the attacks of pain with vasomotor and trophic changes. It is not astonishing that syringomyelia was thought of. The dissociation of sensation, impairment of pain and temperature senses with greater integrity of tactile sense, was like the disturbance of sensation in syringomyelia, and in 1889 syringomyelia was not so well understood as in 1901. We now know that dissociation of sensation may occur in tabes.

---

[1] Traité de Méd., vol. vi. p. 415.

The condition known among the Germans as "springing pupils" ("springende Pupillen"), or "springing mydriasis," deserves attention. Koenig[1] says it is of rare occurrence, and comparatively little has been written on the subject. It has been observed in paretic dementia and tabes, and regarded as a bad omen, but is not necessarily so. It has preceded most of the other signs of paretic dementia five or twelve years (Mendel, Hirschberg). It consists of mydriasis affecting first one eye and then the other, and while it may be found in persons without organic nervous diseases, it should at least make the diagnostician guarded in his statements regarding prognosis. Riegel[2] also believes that it is not necessarily a bad sign. In Dr. Cohen's case the sign was very prominent, and was observed among the first manifestations of disease.

I have not found any reported cases of cervical tabes in which the alteration was in the early stages of the disease, as in this case I have studied, and which so far as the spinal cord was concerned was not only a case of tabes superior, but also one of tabes insipiens. The nerve fibres were not all diseased even in those posterior roots that were most altered, and consequently degeneration of the columns of Burdach is not very intense, because normal fibres in these columns are so intimately mingled with the degenerated fibres that sclerosis has not become prominent, and yet the degeneration was not recent, as no evidence of recent alteration was obtained by Marchi's method. For some mysterious reason the disease of the posterior root fibres progressed very slowly, and seems to have been almost arrested for years, as we may judge from the slow progress of the clinical symptoms and the slow death of the posterior root fibres.

A. Stcherbak[3] is correct when he says we must assume that in the early stages of tabes all the root fibres in the various segments of the cord are not equally affected. The case I have examined permits more than an assumption, and shows that there is a very incomplete degeneration of any one posterior root. It is because of this fact that there are many variations of the clinical phenomena; possibly the fibres conducting the sensory impressions from the skin may be first affected or those from deeper structures, so that in the early stages of tabes there may be disturbance of the cutaneous sensation alone, or loss of the deep sensation without disturbance of cutaneous sensation, or ataxia without disturbance of sensation.

---

[1] Deutsch Zeitschrift für Nervenheilkunde, vol. xv., Nos. 1 and 2, p. 122.

[2] Ibid., vol. xvii., Nos. 1 and 2, p. 169.

[3] Neurologisches Centralblatt, 1900, No. 23.

The cases of cervical tabes with necropsy, so far as I have been able to collect them from the literature, are those of Leyden, Dejerine, Martius, Eichhorst, Marinesco, Redlich, and probably those of Mayer and Vucetie. The name of tabes cervicalis seems to have been first employed by the elder Remak for that form of tabes in which the upper limbs are chiefly implicated. It is a very rare type of the disease, and in 106 cases of tabes studied at the Bicêtre, Dejerine[1] observed it only once. It is known also as tabes superior, and on account of its resemblance clinically to syringomyelia Marie[2] warns us to be careful in the differential diagnosis. Very few cases have been reported in the literature, and if we include only those with necropsy the number is exceedingly small. The only cases of tabes cervicalis referred to by Leyden and Goldscheider,[3] in their recent work in the Nothnagel system of monographs, are those of Leyden, Martius, Dejerine, and Redlich. Redlich,[4] in his monograph on tabes, in which we should expect to find the literature fully given, refers to the cases of Leyden, Martius, Eichhorst, Dejerine, Vucetie, Marie, Mayer, and Marinesco. In an earlier paper on tabes, Redlich[5] described a case of cervical tabes, and in his monograph already referred to he describes another. It is desirable to collect these cases of cervical tabes so far as possible, and to compare the symptomatology. In the few that are reported with necropsy the degeneration of the posterior roots does not seem to have been confined to the cervical region, so that in a strict sense tabes cervicalis is a misnomer, but it has been long in use and the predominance of the symptoms is the result of cervical implication, and it is therefore likely that the term will not be abandoned.

In 1876, in his "Klinik der Rückenmarkskrankheiten," Leyden[6] said: "In rare cases the disease begins in the upper limbs; the pains and sensory disturbances are felt here first, and remain most severe here. The lower limbs during the entire course of the disease are not much affected. A case of this kind was described by Gull, and I have recently observed a similar one. The recognition of a case of this character is more difficult because the ataxia is not so prominent. . . . In other cases the symptoms indicate an implication first, or at least chiefly, of the upper portion of the spinal cord; the pains in the head, face, and neck are severe, the ocular symptoms develop early, and the pains of the lower extremities diminish and ataxia does not develop until late." The findings in Leyden's case, to which Leyden[1] refers briefly in the above quotation, he said later were very similar to those in Martius' case. The columns of Burdach were the ones chiefly degenerated, while those of Goll were very slightly affected. Leyden, in 1888, said that his own case and Martius' were the only cases of tabes cervicalis with microscopical examination of the spinal cord on record. It will be seen from this that accurate knowledge concerning the symptomatology and pathology of tabes cervicalis dates from 1876, and yet in the past twenty-five years very few cases with necropsy have been reported. I have not been able to obtain the papers containing the reports of the cases of Mayer and Vucetie.

---

[1] Sémiologie du Système Nerveux. Traité de Path. Gén., vol. v. p, 650.

[2] Vorlesungen über die krankheiten des Rückenmarkes, translated from the French, p. 325.

[3] Nothnagel's Specielle Pathologie und Therapie, vol. x. p. 508.

[4] Die Pathologie der tabischen Hinterstrangserkrankung, 1897.

[5] Obersteiner's Arbeiten, 1892, vol. i. p. 26.

[6] Klinik der Ruckenmarkskrankheiten, zweite Band, zweite Abtheilung, p. 350.

In 1888 four cases of cervical tabes, two of which were with necropsy, were published. They were those of Weir Mitchell, Dejerine, Martius, and Bernhardt. Weir Mitchell[2] described a case of cervical tabes without necrospy in which the knee-jerks were excessive on each side.

In Dejerine's[3] case of superior tabes the disease began with impairment of vision and diplopia. Both optic nerves became atrophied and myosis was present. Lancinating pains were felt in the upper limbs and in the back part of the head and in the trunk and thighs. The movements of the upper limbs became so impaired from ataxia that the man had little use of those parts. Walking was difficult, simply because he could not see well, but the movements of the lower limbs were not ataxic. Tactile and pain senses, but not temperature sense, were much retarded in the upper limbs and face, and less so in the trunk. Sensation was almost normal in the lower limbs. The muscular sense was lost in the upper limbs, but preserved in the lower limbs. The olecranon reflex was lost, but the patellar reflex was preserved in the adductors of the thighs, although the jerk of the knee was absent. Romberg's sign was not present. Taste, smell, and hearing were preserved. No visceral disturbances existed. The cerebrum and cerebellum were found to be normal, the posterior roots were much degenerated in almost the entire cervical region and upper part of the thoracic region, and very slightly so in the lumbar region. Slight posterior leptomeningitis existed. The anterior roots were normal. The spinal root of the fifth nerve was much degenerated on each side. Both sixth nerves were degenerated. The posterior columns were much degenerated in the cervical region, less so in the mid-thoracic region, and very slightly so in the lumbar region. Slight alteration of Clarke's column was observed. The anterior part of the spinal cord and the nuclei of the cranial nerves were normal.

---

[1] Deutsch med. Wochenschrift, 1888, vol. xiv., No. 9, p. 164.

[2] Medical News, April 21, 1888.

[3] Archives de Physiologie, 1888, 4 Série, Tome I. p. 331.

In the case of cervical tabes reported by Martius the disease began with paræsthesia in the upper limbs, especially in the hands and fingers, and later similar paræsthesia was experienced in the lower limbs, with weakness of those parts. Ataxia could not be detected. The man was unable to distinguish objects by touch when his eyes were closed, and he had some disturbance of pressure and temperature senses. Romberg's sign, Argyll-Robertson's sign, and Westphal's sign were absent; indeed, the knee-jerk was easily obtained on each side and without reinforcement. Still later girdle sensation was felt. The patient died from pneumonia in the same year that his symptoms were studied, but during the pneumonia reflex rigidity of the iris was detected. The knee-jerk was present until death. The degeneration of the posterior columns was most intense in the cervical region, and was very slight in the lumbar region, and Westphal's root entrance zone was fully intact.[1]

Bernhardt's[2] case of cervical tabes was without necropsy. The knee-jerks were absent and the ataxia was very pronounced in the upper limbs but not in the lower.

In Eichhorst's[3] case the posterior columns were degenerated in the cervical and upper two-thirds of the thoracic region. The posterior columns in the lower third of the thoracic region and in the lumbar and sacral portions were not affected, and yet the knee-jerks were lost. Leyden believed that loss of the patellar reflex might occur in tabes from neuritis, but Eichhorst was the first to actually demonstrate in the report of this case that the loss may be so produced. He found intense neuritis of the crural nerves. It is interesting to know that while in this case the posterior roots of the lumbar region were not degenerated, the more peripheral parts of these neurons were greatly altered.

In the case of superior tabes reported by Marinesco the degeneration in the columns of Burdach was distinct at the third cervical segment, and on one side the posterior roots were degenerated as high as this level. The degeneration of the posterior roots extended downward as far as the seventh thoracic segment. The posterior roots were not altered in the lumbar region, but in the cervical the degeneration of the columns of Burdach was intense, much more so than in the case I have studied. The patient had had ocular palsies, impairment of vision, pain in the right shoulder, partial paralysis of the right upper limb with anæsthesia and analgesia of this limb extending to the thorax on the same side, masque tabétique (Charcot), and loss of sensation in the conjunctiva and cornea. There was no disturbance of the muscular sense. The patellar reflex was preserved. The pupils were equal, somewhat contracted, and the irides did not react to light or accommodation. The visual fields were contracted. The oculomotor nerve was paralyzed on each side. The bulb does not appear to have been examined. In the atlas in which this case is published Marinesco[1] gives a picture of a section from the cervical cord from another case of cervical tabes, and in this case the tabetic degeneration was in an early stage.

---

[1] Deutsch med. Woch., 1888, No. 14, p. 163.

[2] Zeitschrift für klin. Med., 1888.

[3] Virchow's Archiv, 1891, vol. cxxv. p. 25.

In both the cases reported by Redlich[2] the degeneration of the posterior roots was not confined to the cervical region. In his second case the "local tabetic degeneration"—by which he refers to the degeneration of the posterior roots at their entrance into the spinal cord—began in the upper part of the thoracic cord and extended as high as the upper cervical region.

In the case diagnosticated as one of cervical tabes by de Buck,[3] weakness, ataxia, lancinating pains, and objective disturbances of sensation were present in the upper limbs. The gait was slightly ataxic and Romberg's sign was present. The Achilles reflex was absent on each side, but the patellar reflex was exaggerated on each side. I am not aware that any case of cervical tabes with exaggerated knee-jerks and necropsy has ever been reported, so that we are in complete ignorance of the cause of this exaggeration. De Buck's case was merely a clinical one.

It is not to be supposed that I have included all the clinical cases of cervical tabes. Some are alluded to in a cursory manner. Mott,[4] for instance, refers to one of his cases in which the cervical tabes began in the upper limbs of a mounted policeman, and infers that the greater use of the upper limbs was the cause of this unusual involvement.

Dr. Cohen's case was one of bulbar tabes as well as one of cervical tabes. Bulbar disturbances as a part of the tabetic symptom-complex are not common, although paralysis of the ocular muscles, either transitory or persistent, has been observed not infrequently. Leyden and Goldscheider[5] have found that paralysis of the external ocular muscles occurs in about 40 to 50 per cent. of the cases of tabes, and that paralysis of the external rectus is the most common of these. In Dr. Cohen's case there was distinct paresis of the right external rectus and partial degeneration of the extramedullary portion of the right sixth root near the medulla oblongata. Paralysis of the facial or of the motor portion of the trigeminus is rare, although Peterson and Schultze have each reported a case of paralysis and atrophy of the muscles of mastication in tabes. Leyden,[6] in 1876, said that unilateral facial paralysis sometimes occurs in tabes, but is incomplete and transitory. Hirt[1] regarded facial palsy as such a rare sign of tabes that when it occurs he thought one should consider carefully whether it were not a complication. He observed it in only two out of 345 cases of tabes. Chvostek,[2] in reporting a case of tabes with sensory and motor disturbances in the distribution of the trifacial nerve, difficulty in deglutition, palsies of external ocular muscles, and paralysis of the right facial nerve in the lower part of its distribution, refers in refutation of Hirt's opinion to the cases of Fournier, Kahler, Semon, Dejerine, Peterson, Jeoffroy-Hanot, and Ehrenberg.

---

[1] Atlas der Pathologischen Histologie des Nervensystems. Victor Babes. v. Lieferung, 1896.

[2] Die Pathologie der tabischen Hinterstrangserkrankung. 1897. Obersteiner's Arbeiten, 1892, vol. i. p. 26.

[3] Journal de Neurologie, June 20, 1899, No. 13, p. 241.

[4] Lancet, July 14, 1900, p. 87.

[5] Nothnagel's specielle Pathologie und Therapie, vol. x. p. 532.

[6] Klinik der Ruckenmarkskranheiten, zweite Band, p. 344.

In all three of the cases of tabes with bulbar symptoms reported by Cassirer and Schiff disturbances in the lower distribution of the facial nerve were observed, but there were no central changes to explain these symptoms.

In Peterson's case of tabes the weakness and atrophy of the masseter and temporal muscles were pronounced, and in eating the man had to support and assist the lower jaw with his hands. The two pterygoids on each side were paretic. There was no anæsthesia of the face. The left side of the face was a little paretic, the action of the occipitofrontalis was stronger and the nasolabial fold deeper on the right than on the left side.[3]

The facial implication seems, therefore, to have been slight. In Dr. Cohen's case the findings in the seventh nerves and their centres do not afford a satisfactory explanation of the facial paresis, and it is possible that it was the result of disturbed muscular sense from degeneration of the fifth nerves.

Hemiatrophy of the tongue in tabes seems to have been first observed by Charcot and later by G. Ballet. When Koch and P. Marie[4] reported their case they could find only five other cases of hemiatrophy of the tongue occurring in tabes, and only two others with microscopical examination (Raymond and Artaud, Westphal, Koch and Marie). In these three cases the twelfth nucleus on the side corresponding to the atrophied side of the tongue was degenerated. Marie later refers to a case with necropsy reported by Mathias Duval. Cassirer and Schiff refer only to the cases of Raymond and Artaud, Koch and Marie, Westphal, and Eisenlohr, as cases of tabes in which either unilateral or bilateral degeneration of the nucleus of the hypoglossal nerve had occurred, and in all these cases except Eisenlohr's the atrophy of the hypoglossal nucleus was unilateral. In a case of tabes reported by Cassirer and Schiff the hypoglossal nuclei in the greater portion of their extent were normal, but in the distal portion of the nucleus on the side corresponding to the atrophy of the tongue there was a very slight diminution in the number and size of the nerve cell bodies. They regarded this alteration as insufficient to explain the atrophy of the tongue, and they believed the atrophy of the tongue was produced by degeneration of the hypoglossal nerve. In the case I have examined the hypoglossal nucleus in its inferior portion on the side corresponding to the half of the tongue at first most atrophied was much smaller than the corresponding nucleus of the other side, and Cassirer and Schiff's observation may have more importance than they attribute to it.

---

[1] Pathologie und Therapie der Nervenkrankheiten, second edition, p. 529.

[2] Neurologisches Centralblatt, 1893, No. 12, p. 762

[3] Journal of Nervous and Mental Disease, 1890, vol. xvii. p. 450.

[4] Revue de Médecine, 1888, vol. viii. Vorlesungen über die Krankheiten des Rückenmarkes, p. 283.

It is my belief that if the medulla oblongata were studied more frequently in cases of tabes, degeneration of the spinal root of the trigeminal nerve would not be a very rare finding. I have seen it in a number of cases of tabes. According to Cassirer and Schiff, the degeneration of this root was first detected by Westphal in 1864, and later by Oppenheim and Siemerling, Hayem, Flechsig, Vulpian, and others. Cassirer and Schiff found the spinal root of the trigeminus degenerated in three cases of tabes, and in one of these the degeneration was unilateral. They point out that in some cases the root was more degenerated in its inferior than in its superior portion (Oppenheim and Siemerling, Eisenlohr, Zeri), and that in others the dorsal part of the root or the ventral part was most degenerated. In the case I have studied as well as in those of Cassirer and Schiff the glia cells seemed to be more numerous in the degenerated spinal roots. In their cases the sensory nucleus of the trigeminal nerve was perfectly normal, as it was at least on one side in my sections, although in cases reported by others it has been found degenerated. The sensory disturbances in the face that have been observed in cases in which the spinal root of the fifth nerve was found degenerated were not always the same, but Cassirer and Schiff say that in their cases sensory disturbances were always present in the face when the spinal root was greatly degenerated, although a parallelism between the clinical signs and the pathological findings was not present. We shall probably not err if we attribute to the degeneration of the spinal root of each fifth nerve, in Dr. Cohen's patient, the disturbance of sensation in the face, possibly the loss of taste on the anterior portion of the tongue, and even at least some of the motor bulbar symptoms, explaining these as a result of loss of sensory impulses.

In one of his cases of tabes, Oppenheim[1] says paræsthesia and disturbance of function were observed in the distribution of the fifth nerve. The patient's face felt stiff, and he had paræsthesia in the mouth and tongue and difficulty in chewing and swallowing, and yet this difficulty was not caused by disturbance of motor nerves or, at most, very slightly so, as the muscles of mastication were powerful and paresis of the soft palate was very slight. Ataxia of the muscles of the face and tongue and of mastication became so great that when the patient attempted to speak he showed great contortion of the face. All these disturbances were probably the result of alteration of the fifth nerve. In another case in which implication of the fifth nerve existed, chewing and swallowing were difficult, the patient did not know whether any food were in her mouth, and she could extend the tongue properly only when she watched the movements of the tongue in a looking-glass. In both these cases the spinal root of the fifth nerve was degenerated on each side.

---

[1] Arch. für Psychiatrie, 1888, vol. xx. p. 131.

Oppenheim[1] says that Duchenne spoke of implication of the fifth nerve in tabes. He also says that the symptoms attributed to degeneration of the spinal root of the fifth nerve have varied considerably in different cases.

The constant biting of the cheeks, for the relief of which in 1886 Dr. Cohen's patient had several teeth extracted, and the absence of pain upon their removal, as well as the loss of food in the mouth, were probably the result of degeneration of the spinal roots of the fifth nerves.

The descending root of the vago-glossopharyngeus is occasionally found degenerated in tabes. Cassirer and Schiff collected twelve cases in which degeneration of this bundle was observed, but only in six of these was the microscopical examination thorough. The absence of disturbance of taste in these six thoroughly studied cases seems to show that integrity of the solitary bundle is not necessary for the function of taste. Disturbances of the functions of the larynx observed in these six cases were not necessarily the result of degeneration of the solitary bundle, because there was also degeneration of the bulbar nuclei or of the peripheral portions of the vagus and glossopharyngeus nerves. In one of the twelve cases the solitary bundle was degenerated, but clinical signs indicative of this degeneration had not been observed. Cassirer and Schiff conclude that the solitary bundle has no relation to the function of taste, or of sensation or motion in the larynx, or to deglutition. The degeneration of this bundle has no relation to gastric crises or disturbances of respiration. Cassirer and Schiff found the large fibres in the solitary bundle degenerated, while the fine fibres were preserved and formed a group on the dorso-median portion of the bundle. In Schlesinger's[2] case both descending glossopharyngeal roots were degenerated, and, according to Schlesinger, the degeneration of these roots has been found in tabes especially when laryngeal disturbances had been present, and yet he merely calls attention to the frequency of the association without expressing himself very positively in regard to the importance of it. It seems from his investigations, also, that degeneration of these roots does not cause disturbance of taste.

---

[2] Wiener klin. Wochen., 1894, No. 27, p. 497.

It is possible, however, that the feeble and irregular cardiac action in Dr. Cohen's patient was the result of the degeneration of the descending root of the left vago-glossopharyngeus.

The dorsal nucleus of the vagus has been found degenerated in tabes, and was so in one of Cassirer and Schiff's cases, but these authors were unable to observe positive degeneration of the nucleus ambiguus, and I also have been unable so to do in the sections studied by me.

Cases of bulbar tabes with necropsy are very rare, and v. Reusz,[1] in publishing a case of this kind, says only two other such cases have been reported since the appearance of the paper by Cassirer and Schiff, viz., a case by Grabower and Oppenheim and one by Zeri, but the case by Zeri is probably referred to by Cassirer and Schiff.

SOME OBSERVATIONS ON TYPHOID FEVER COMPLICATED BY CROUPOUS PNEUMONIA, WITH REPORTS OF FOUR FATAL CASES.

By Henry M. Fisher, M.D.,
of Philadelphia.

Many text-books do not even mention croupous pneumonia as a complication of typhoid fever.

As Niemeyer says,[2] it can easily be overlooked in the same way as the pleurisy with effusion that also occasionally occurs as a complication of typhoid, and it, like the latter, is often unattended either with pain, chill, or increased fever.

That it is not a very unusual complication is shown by the study of Liebermeister's[3] cases in the epidemic of typhoid fever at Basle, 1865–1868, when consolidation of the lungs, not due to hypostatic congestion, was noted fifty-two times among 1420 cases, giving a percentage of the complication of 3.6. That it is a very serious complication is shown by the fact that of these fifty-two cases reported by Liebermeister[4] and Betke,[5] twenty-nine died, showing a mortality, therefore, of 55.7 per cent. Gallisart de Marignac[6] reports thirteen cases with only three recoveries, a mortality for the complication, therefore, of 77 per cent.

Other French observers, as L. Girard,[7] Castex,[8] and G. Destaix,[9] all report a number of cases with a low percentage of mortality. I think,

---

[1] Archiv f. Psychiatrie, 1899, vol. xxxii., No. 2, p. 535.

[2] Lehrbuch der speciellen Pathologie und Therapie.

[3] Ziemssen's Cyclopædia.

[4] Loc. cit.

[5] Die Complicationen des Abdominal Typhus. Berlin, 1870.

[6] Pneumonie lobaire. Survenant dans le cours de la Fièvre Typhoïde. Paris, 1877.

[7] De la fièvre typhoïde a début pneumatique. Paris, 1882.

[8] Contribution à l'etude des accidents pulmonaires de la fièvre typhoïde. Paris, 1879.

[9] Considerations sur quelques accidents pulmonaires, et sur la pneumonie pseudolombaire en particulier, survenant dans le cours de la fièvre typhoïde.